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Technical Note: Stafne Static Mandibular Bone Defect—Further Expression on the Buccal Aspect of the Ramus

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ABSTRACT The anatomic distribution of the Stafne static mandibular bone defect (SSBD) is extended with a description of a cavitation defect on the buccal ramus of the mandible. The anatomical placement of SSBD thus correlates precisely with the submandibular and parotid salivary glands, and gives further evidence that an increase in major salivary gland size is associated with the defect. The global latitudinal variation in the population prevalence of SSBD ranges from 10% in the tropics to virtually 0% in most of the arctic. Globally the defect directly correlates with parasite load and diversity, and may be a marker for of a history of an environment with high levels of enteric macroparasite infestation. Am J Phys Anthropol 111:425–427, 2000.

Stafne static mandibular bone defect (SSBD) is a singular skeletal variant with a highly unusual and interesting history. The defect is singular in the sense that it is the only chronic active bone resorbing and yet static lesion of which I am aware. That is, at the base of the defect it actively resorbs and forms bone at the same time, and yet after its initial appearance, the defect does not progress in size over time, neither surface area, nor in depth (Shields and Mann, 1996). Data were presented suggesting that SSBD is etiologically heterogeneous (Shields and Mann, 1996). The putative cause of the defect is either an enlargement of the submandibular and parotid glands or their fixation, causing their chronic tight apposition to the mandible. Under this "glandular" hypothesis, a narrow gradient of a salivary gland-produced osteolytic factor that is normally "leaked" from the major salivary glands (MSG), and causes no effect, is able to produce a highly localized persistent resorption of contiguous bone. Only through this chronic apposition does a defect arise. The bone reacts to emitted osteolytic factors with bone formation, thus creating a chronic stationary front that is nonetheless, dynamic. Biologically active factors produced by the MSGs (Shields, 1998), and further biological and structural aspects of MSGs are summarized, for example, by Dobrosielski-Vergona (1993).

Intriguingly, great variance in the global latitudinal prevalence of SSBD was shown to steadily decrease from 10% in the tropics, 4% in the temperate zone, to 1% in the arctic (Shields, 1998). In this brief communication, a further clinical manifestation of SSBD is described on the buccal aspect of the mandibular ramus. This observation is consistent with the hypothesis that SSBD is a MSG-related character not restricted to the region of the submandibular salivary gland. SSBD does not cause a clinical problem, although it can be misdiagnosed as an ominous lesion.

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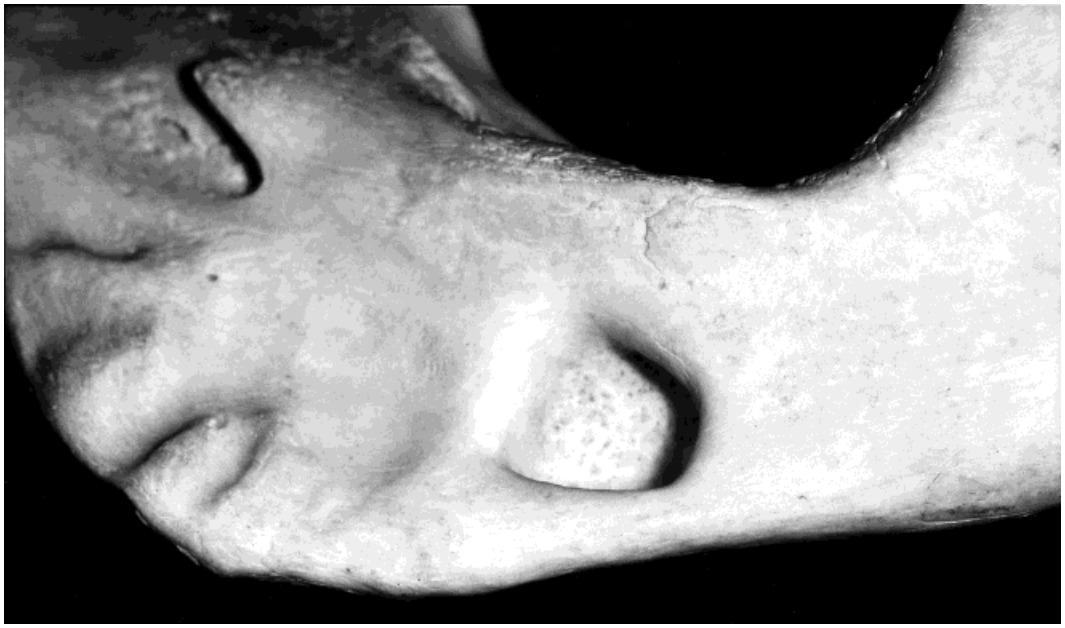

Fig. 1. A large Stafne static mandibular bone defect in the submandibular fossa of the body of the mandible.

MATERIALS AND METHODS

From human populations sampled globally within the past 2,000 years (acknowledging the nature of ascertainment of museum skeletal material), 7,686 mandibles were examined for SSBDs (3,876 females and 3,810 males). The mandibles were housed in the following museums: National Museum of Natural History, Smithsonian Institution (Washington, D.C.); American Museum of Natural History (New York); Field Museum (Chicago); Peabody Museum, Harvard University; the Museum of Civilization (Ottawa), and the British Museum of Natural History. The SSBD described here was found on the right mandibular ramus in a skeletal specimen of a male from "Gold Coast" West Africa (Ghana) (American Museum of Natural History #VL5274).

RESULTS AND DISCUSSION

The original description of SSBDs identified their highly restricted anatomic distribution. These are seen only below the mylohyoid ridge in the submandibular fossa of the mandible (Fig. 1) (Stafne, 1958). Wolf (1985), and Mann and Shields (1992) extended the anatomical distribution of the defect by describing SSBD on the lingual ramus, which suggested that the parotid gland is also associated with SSBD. Defects on the lingual ramus represent 8% of all described defects and are as frequent as bilateral defects of the body of the mandible (Mann and Shields, 1992).

In Figure 2, a SSBD is seen on the right buccal ramus. It measured 8.5 mm in width, 4.7 mm in height, and 0.3 mm in depth (overall SSBD: n = 175, $\bar{x}$ length = 6.9 $\pm$ 3.4 mm, $\bar{x}$ height = 4.8 $\pm$ 2.2 mm, $\bar{x}$ depth = 1.6 $\pm$ 1.2 mm). The shape of the defect is not only statistically average for a SSBD, but its subjective clinical appearance is also typical for SSBD (Fig. 2). The periphery the defect has a gently rolled border of smooth cortical bone that at its base sharply meets exposed trabecular bone.

This is the first description of a SSBD on the buccal aspect of the mandibular ramus. The mandibular ramus is contiguous with aspects of the parotid gland which folds from the lingual surface of the ramus onto the buccal aspect of the condylar process between the temporomandibular ligament and

Fig. 2. A Stafne static mandibular bone defect on the buccal aspect of the ramus just to the right of the number "5274." Varnish covers the lower third of the defect.

the masseter. Thus any surface of the mandible which is potentially chronically exposed to a major salivary gland can express SSBD.

The worldwide prevalence of SSBD is highly patterned among global isotherms with an exponential decrease in male prevalence extending from the tropics, to the arctic (Shields and Mann, 1996). This latitudinal structured decrease in prevalence was observed regardless of ethnic background. Such a global distribution implies strong environmentally related forces of selection in the pathology of SSBD. An attempt to explain this highly patterned distribution is the suggestion (Shields, 1998) that SSBD is a sign of enlarged major salivary glands, and that the major salivary glands are a focus of selection. Larger major salivary glands produce more biologically active factors. Increased amounts of salivary gland-secreted natural immunity factors targeted to the alimentary system would enhance protection against enteric macroparasites. Especially in the tropics, a burden of gut macroparasites frequently persists even in communities with low population density. Many founder populations were likely composed of a small number of individuals. The hypothesis that potentially powerful forces of selection acted on aspects of major salivary gland physiology needs to be rigorously tested.

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